

Online Journal of Health Ethics

Volume 14 | Issue 1

Article 9

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Recommended Citation

Andrade, G. (2018). Should Doctors Take Into Account Human Races? A Medical Ethics Approach. *Online Journal of Health Ethics*, 14(1). <http://dx.doi.org/10.18785/ojhe.1401.09>

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Should Doctors Take Into Account Human Races?

A Medical Ethics Approach

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ABSTRACT

Racial determinations that lead to race-based treatments and mistreatments have many harmful social effects. When used in the practice of medicine, can racial determinants lead to good outcomes? This is an emerging question in medical ethics. It is undoubtedly true that some individuals are more genetically prone to some diseases than others. However, should one rush to judgment with the belief that race may be a valid indicator in identifying diseases an individual is susceptible to? Furthermore, should race be considered in prescribing treatment? Illnesses such as sickle cell-anemia and Tay Sachs disease have long been thought to have a racial origin. This assumption is challenged in this paper. There have also been attempts to prescribe specific drugs for specific racial groups; but this is approach call for further inquiry. Belief in the presupposition that disease is a bio-psycho-social process implies that genetic predispositions are only one factor among many others that relate to the way things are socially constructed. This paper seeks to critique one's view of the use of race as a criterion of medicine to prescribe treatment.

Key Words: Race, Medicine, Medical Discrimination, Disease, Health, Medical Ethics, Racial Determinants

Editor's Note: Kindly consider viewing Dr. Dorothy Robert's *Ted Talk: Race Based Medicine as a follow-up to Andrade's article*:

https://www.ted.com/talks/dorothy_roberts_the_problem_with_race_based_medicine

Also consider viewing Dr. David William's *Ted Talk: How Racism Makes Us Sick*

https://www.ted.com/talks/david_r_williams_how_racism_makes_us_.

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1. Introduction

In the wars that took place between English settlers and American Indians in the eighteenth century, there was a very unfortunate episode. While British settlers were besieging Fort Pitt (in the state of Pennsylvania, in the US), Jeffrey Amherst is said to have derived the idea of giving the Indians blankets infected with smallpox. Amherst reasoned smallpox would decimate the indigenous population, and so end the siege of the fort and the conflict in general (Pedersen, 2010: 71).

History remains conflicted on the actual implementation of Amherst's plan, but he has remained in infamy as one of the pioneers of biological warfare. If, indeed, he employed such a deliberate tactic, it may have killed half a million people. Whether it was deliberate or not, the fact is that smallpox nearly annihilated the indigenous peoples of America (Parker, Schutz, Meyer and Buller, 2010: 467). Often Spanish conquistadors are accused of genocide, but in reality, most of the conquest of the Americas was done with microbes. A few centuries before the arrival of Columbus to America, terrifying plagues razed at least one third of the European population.

When Europeans came to America, they brought these diseases. It did not constitute a serious threat to Europeans. But for indigenous people it was fatal because they had never been in contact with them. They did not have a sufficiently strong immunity. Anthropologist Jared Diamond (1999) suggested that the absence of domesticated animals in the Americas gave Native Americans little contact with germs, and this made them more likely to succumb to European diseases: smallpox, influenza, measles, etc. The result was tens of millions died in one of the most terrible epidemics humanity has ever known.

One of the lessons of this tragic story is that some populations of the world are more vulnerable than others to certain diseases. And in the same manner that amongst populations there are varieties in height, skin color or frequency in the production of lactase, there are also differences in genes encoding resistance to different types of microbes. In the context of the conquest of America, smallpox became a disease afflicting much more the natives (Robertson, 2001).

Thus, vulnerability to certain diseases can be used as a criterion for segmenting humanity in different populations. And in the same way that some pseudoscientists have tried to establish a correlation between skin color and other racial traits, as it was typical in the nineteenth century during the heyday of racial science, it has also been postulated that there is a correlation between racial traits and certain specific diseases.

Under this argument, select diseases are said to have a genetic basis. This is similar to the opinion of some racialists that darker skin has a correlation with genes, which encode with lower intelligence. As such, some racialists posit that racial traits are correlated with genes encoding increased vulnerability to certain diseases

Thus, a catalog of racial diseases has been produced. Cystic fibrosis afflicts particularly whites (Hopkin, 2010). Sickle cell anemia is a disease of blacks (Hill, 2003). Tay-Sachs disease is almost exclusively Jewish (Walker, 2007). Tuberculosis incidence is highest among Native Americans (Young, 1994). And, Gypsies have a disproportionate percentage of patients with asthma (Greenfields, Darlymple and Fanning, 2011: 215). And so it goes with many other diseases.

Unlike what happens in other spheres of social life, these racial labels supposedly serve a laudable purpose: to make medical practice more effective. While postulating that blacks are more inclined to crime is extremely destruction in that it promotes police abuse; but, to postulate that blacks are much more vulnerable to sickle cell anemia, so the argument goes, is supposed to be of great help. Consider, if a doctor sees a black patient with some symptoms of this disease, it is supposed to be easier to make a diagnosis (inasmuch as, given the racial profiling, it will be easier to rule out other diseases), and attack the disease in quicker manner.

According to this argument, physicians should not only consider race when developing diagnoses, but also administering drugs. The logic is conferred that racial groups also have different responses to drugs.

2. The prospect of personalized medicine

Pharmacogenomics technologies, although in embryonic stages, offer the prospect of being able to analyze the genome of a person and encode reactions to different drugs. From this information, one should be able to make the determination of which pharmacological agent will work best, and the dose to be administered.

These technologies are very promising, and if developed, would be a tremendous medical breakthrough. Pharmacogenomics could offer the possibility of personalized medicine. Each physician and other healthcare provider (i.e. nurse practitioners and physician assistants) would indicate a detailed dose on the basis of genetic information of each patient, and this would make more effective treatments (Innocenti, 2005).

Certainly, individualized medicine and dosing would be an ideal scenario. But, we are still far from it. Until science finishes sequencing the complete genome of individuals, we are very far from being able to follow this strategy. And, sequencing the genome is just a first step; then comes the (much more difficult) procedure of specifying in the genes different reactions to drugs.

Therefore, for now, personalized medicine is more a matter of science fiction than real medical practice. However, in the meantime, there have been doctors who have postulated that although we cannot yet offer individualized medical care, racial groups can at least guide us. In that sense, if we have data that allow us to assume that certain racial groups react better to some drugs and not others, then we can establish a specialized racial allocation of some medications.

Psychiatrist Sally Satel, for example, admits that in surgery to repair a broken leg, the patient's race is irrelevant. But when administering Fluoxetine, Satel chooses to prescribe higher doses at baseline to white patients, and lower doses to black patients. According to Satel, black patients metabolize antidepressants more slowly than white or Asian patients, and thus, a high dose increases the risk of adverse reactions (Satel, 2008).

This racial discrimination in medicine is not exclusive of psychiatry. For years there has been discussion about the possibility of drugs targeting specific races. And, in 2006, there was on the market a drug intended only for black patients, BiDil (Pollock, 2012: 165).

This drug is applied to treat heart disease. In the 1980s, tests were made with this drug, and it was administered to different groups without a significant result. However, blacks did demonstrate a significant improvement. Two decades later, tests were done only with members of the black race, and positive results were documented in the application of the drug. So, after these tests, US health authorities approved BiDil on the market, only to be prescribed exclusively to blacks.

BiDil did not work well in the pharmaceutical market. But, it opened the door to consider formulating drugs specifically aimed at racial groups. And, predictably, it has sparked a controversy. Certainly the idea that there are human races has been very harmful, but can this idea be used now to save lives? Should doctors take into account human races?

3. Are there racial diseases?

The idea that there are diseases associated with specific races, and if this proposition is true, that we can discriminate racially in the application of drugs, remains ambiguous. First, it is doubtful that racial segmentation of diseases and their treatments are always done with the laudable purpose of saving more lives. The practice of medicine is a double-edged sword. Much good can be done, but medicine can also serve to legitimize many abuses. And, as doctors often have the power to save lives, they also have the power to strip freedoms to some groups in specific, all in the name of public health. When a racial group is labeled with a specific disease, the compass is opened for the social system, sponsored by the medical establishment, to impose greater discrimination on a given particular group.

A racial group labeled with a disease can easily become a public menace, as it allegedly has the danger of spreading the disease to the rest of society. If it's not a contagious disease, it has the danger of mixing their harmful genes with the rest of society through miscegenation, and thus give rise to a sick generation. Or, the racial group in question can become a heavy burden on the public health system because of its increased vulnerability to certain diseases.

Departing from this, it is much easier to justify segregation systems as a kind of permanent quarantine; or prohibit marriage between members of different racial groups in order to prevent a race to become contaminated with harmful genes from the other; or to forbid immigration in order to prevent immigrants from bringing their diseases and make the public health system collapse.

Sickle cell anemia is a good example of how racial profiling in medicine can bring dire social consequences. Since the beginning of the twentieth century in the US, the idea that this disease afflicts blacks exclusively came up. Before the civil rights movement in the 1960s, in the southern states, there was a terrible system of racial segregation sanctioned by law (not unlike the South African apartheid). Under that system, each individual was assigned a racial category, and this racial categorization determined who could occupy seats on a bus, where they could eat, whom they could marry, etc.

Under the one-drop-rule, a person was considered 'black', even if most of his or her ancestors were white. It was enough to have one black ancestor, to be considered black. In that sense, there was in the US a considerable segment of the population that, for bureaucratic purposes, was black, but that might look white, and could even circumvent the system of discrimination with their physical appearance. Amongst the dominant white population, there was always a suspicion that some of their members actually were not white, because there was a possibility that they had a black ancestor.

When a person suspected of being black was diagnosed with sickle cell anemia, he or she was definitely assigned to the black race, which was a huge loss of privileges and social prestige. In that sense, the racialization of medicine in that context was not used to save more lives; rather, it served more to strengthen the system of racial discrimination (Wailoo: 2001).

Much more serious was the experimentation that the US government did with blacks suffering from syphilis, from the 1930s onwards. As often happens with AIDS today, at that time there was a widespread belief that syphilis was a disease that especially afflicted blacks, by virtue of their sexual transmission (racialists have come up

with several theories attributing blacks higher levels of sexual activity). Thus, the US government organized the infamous Tuskegee experiments, which consisted in observing black syphilis patients without their consent, to assess how the disease progressed (Uschan, 2006).

It may be objected, of course, that the fact that a few decades ago, the racialization of medicine was used to oppress, does not mean that today, it pursues the same goal. But the fact is that many of the alleged racial diseases are not really such. In many of these diseases some correlations have been established between high incidence of certain diseases, and certain racial characteristics. But here again, the problem appears: correlation is not the same as causation. Perhaps the correlation between skin color and susceptibility to a specific disease, is due to a hidden variable we have not yet considered, and in that sense, it would not be valid to postulate that this or that disease have racial origin.

4. Sickle cell anemia and Tay Sachs disease

Consider again sickle cell anemia. This genetic disease is caused by the inability of red blood cells to carry oxygen, and they acquire a shape like a sickle, instead of the normal form, which is more like a disc. This generates circulatory problems, pulmonary inflammations, cognitive functions, and especially less resistance against infections. Less than 10% of patients who suffer it survive to adulthood.

In the popular imagination, it is a disease of the black race. And so it has been frequently postulated that there must be a close relationship between the genes encoding the typical racial characteristics used to identify blacks, and genes encoding sickle cell anemia. But statistically, this disease is far from being exclusive of blacks. We find significant frequencies in Mediterranean European countries, Arab countries and India. According to conventional racial classifications, these populations belong to different racial groups. So that cannot properly establish a causal relationship between a racial type and the frequency of sickle cell anemia.

We must rather seek other variables. Geneticists have found a very significant variable to explain why sickle cell anemia is most common in those regions. Today we know that two copies of a gene encode this disease. However, when only a copy of that gene is found, that encodes a considerable resistance to malaria (Zack, 2002:54).

Thus, in tropical regions traditionally affected by malaria, there is also a higher frequency of sickle cell anemia. In these regions, it is advantageous to have a copy of the gene in question, while encoding resistance to malaria. That facilitates an increase in the frequency of that gene. But, by increasing the frequency of this gene, the frequency of people born with two copies of the gene is also increased, and thus a higher frequency of sickle cell anemia.

This puts in evidence that sickle cell anemia is not really a racial disease. There is no direct relationship between dark skin and any specific sickness. There is, however, a direct relationship between resistance to malaria and sickle cell anemia, and this has meant that populations in tropical areas with more favorable weather conditions for malaria also develop greater frequency of the allele encoding sickle cell anemia.

For example, in some regions of northern and eastern Africa, where there are not favorable conditions for malaria (regions where the mosquito that transmits the parasite is less abundant), such as in deserts or high altitude areas, the frequency of sickle cell

anemia is very low. Again, this is an indication that the disease is not associated itself with racial traits (as the inhabitants of these areas, especially in eastern Africa, are considered members of the black race), but instead with resistance to malaria.

There have been some racialists that think that the persistence of sickle cell anemia in populations traditionally not considered black actually is due to mixtures with black populations in the past (Tapper, 1999:25). For example, to explain why in Spain (whose population is not considered black) there is a higher frequency of this disease (compared to other European countries), it has been argued that this was due to the introduction of black population during times of Al-Andalus, and even going back to the time of Hannibal! Again, these explanations fail to consider that this disease has a relationship, not with skin color or other racial traits, but with the vulnerability of the Mediterranean region to malaria, especially since the beginnings of agriculture (as it was during this time when, by paving the forests to make way for agricultural fields, the mosquito found a favorable niche).

Tay-Sachs disease is another one that has been commonly racialized. This disease is caused by a genetic mutation that affects the production of an enzyme that regulates the level of lipids in the brain and nervous system. This leads to a neurological impairment, and its symptoms are red spots on the retina, disproportionate head growth and mental retardation, among others. This disease is encoded similarly to sickle cell anemia: to be expressed, there must be two copies of the gene.

Initially, it was reported that the disease afflicted especially Ashkenazi Jews (Jews from Eastern Europe). Then it was postulated that the disease was almost exclusively Jewish, and that it was the result of crossbreeding between relatives (this disease is encoded by a recessive allele, and this type allele is more common in descendants of closer inbreeding).

Some racialists believe that that the high level of intelligence amongst Jews is attributable to their genetic composition, which has resulted from their refusal to interbreed with other populations: racist Kevin MacDonald (2002: 293) even thinks that from early on this was a deliberate Jewish eugenic strategy. Several authors have also postulated that Tay-Sachs disease has a relationship with intelligence (analogous to the relationship between resistance to malaria and sickle cell anemia): two copies of the gene encode Tay-Sachs disease, but a copy of the gene could encode higher levels of intelligence, and that would explain why Ashkenazi Jews are so smart (Oster, 2012).

This comes from a genetic phenomenon called 'heterozygote advantage'. When considering diseases such as sickle cell anemia or Tay-Sachs, an enigma arises: how do these conditions persist in the population? Why has natural selection not definitively destroyed the genes that encode them? An answer has been provided: because these diseases are generated by recessive alleles. Genes encoding these diseases must also encode a variant in some advantage in heterozygous alleles.

In the case of Tay-Sachs disease and others afflicting especially the Jews, it has been postulated that their heterozygote advantage is a higher degree of intelligence. But this seems to be a hasty conclusion. Until now, geneticists have only established Ashkenazi correlation between high frequency of genes for Tay-Sachs (and other diseases), and high level of intelligence. This in no way proves that these same genes encode a high level of intelligence.

Moreover, it is not entirely clear that Tay-Sachs is a "Jewish disease". First, it is difficult to accept that there is a "Jewish race": although there is some debate on this topic, most geneticists agree that Jews are an ethnic group with no particularly important genetic markers. While there are some genetic similarities among Jews, they do not share a number of well-defined biological features that enable them to be labeled a racial group (Corcos, 2005). And, in no way is Tay-Sachs a disease unique to Jews. Today (thanks to genetic testing) it is virtually eradicated among the Jews, and it afflicts more other populations.

5. Special drugs for each race?

Just as it is not entirely appropriate to postulate that there are racial diseases, it is not entirely appropriate to postulate that some drugs should be reserved for specific races. It is worth remembering that the hope of pharmacogenomics is to eventually set up a personalized medicine. While we get to that stage (and, I am optimistic that someday we will get there), some doctors advocate the use of racial categories as a guide to form an idea of which drug works best in individuals. But even Sally Satel (the psychiatrist who admits to offering different drugs to blacks and whites) warns that race is a very rudimentary guide, and that the extent of racial profiling in medicine is very limited.

The case of the drug BiDil is illustrative. BiDil scored well in tests with black patients. However, from the outset these tests were subject to objections, and they were criticized because there was no sufficient control studies with white patients (or other racial groups), in order to isolate the racial variable accordingly (Pollock 2012:165). But also there is another fundamental objection: patients who were part of the study were black, only to the extent that they self-defined as such. In other words, their racial affiliation was conditioned by the social construction that stipulates the rules of who belongs to any given racial group. This is clearly a sampling bias.

It is worth remembering that in the US (the country where BiDil was marketed), traditionally a person is considered 'black' if just one of his or her ancestors is black (the so-called one-drop-rule). Barack Obama is considered the first black US president, but in reality, Obama is the son of a white woman (in Brazil, for example, he would not be considered black), and in addition, his father was a native of East Africa, a region different from the one whence slaves came to America. If someone like Obama had participated in the test BiDil, he would surely have been identified as 'black'. But this could alter the results, because under another social convention (such as the one used in Brazil), Obama may have been well placed in the group of whites, and his positive response to BiDil would have been counted among white patients who react well to this drug, and not among black patients.

That does not mean that racial profiling in medicine has absolutely no value, because perhaps Sally Satel is right when she argues that even if race is a very rudimentary guide to form an idea of the genome of a patient, it is at least one tool preferable to having no guide. Even if he does not accept the existence of human races, commentator Kenan Malik argues that many of the ways in which we group people, are not entirely arbitrary from a biological point of view, and we can have some confidence that members of some racial groups retain more genetic proximity to each other than two randomly selected individuals (Malik, 2009: 37).

But racialized medicine also has risks, and it therefore should be taken with extreme caution. These risks are not merely the dangers of social oppression to which I have already referred. There is also the risk that racialized medicine may cultivate stereotypes that prevent optimal diagnosis and appropriate allocation of drugs, precisely because if there is suspicion that an individual has this or that disease, a racial group may be socially stigmatized with that disease. For example, in the US, there are many documented cases of individuals who have been diagnosed too late with sickle cell anemia (significantly reducing their quality of life), because they did not belong to the black race, and their racial affiliation did not raise suspicion among treating physicians, to take note of symptomatology.

6. Disease as a biopsychosocial process

Sickle cell anemia and Tay Sachs disease are completely determined by genetics. However, there are many other diseases that have only a partial genetic basis, and many environmental variables influence their manifestation and development. However, since it is presumed that these diseases have a genetic basis, it has also been relatively common to attribute them to specific racial groups, because of their genetic makeup.

In the US, for example, it is a common idea that blacks have a genetic predisposition to hypertension, Hispanics are predisposed to diabetes, and Native Indians are predisposed to alcoholism. In Latin America, there is also the idea that Native Indians, by virtue of their genetic makeup, are much more susceptible to tuberculosis than the rest of the population.

Sometimes racialists have formed curious theories about why some groups are more vulnerable than others to certain diseases. In the case of indigenous peoples of America, it has been postulated that their weak immunity against pathogens from Asia and Europe explains why today they suffer disproportionately from diseases such as tuberculosis. Certainly, this theory is adequate to explain why the Native American population in less than a century, was decimated following the arrival of Europeans. But it is doubtful that five centuries later, it serves to explain why Native Americans still suffer from tuberculosis in large numbers (there is also the possibility that tuberculosis was already in America before the arrival of Columbus (Finer: 2009: 14)). Precisely, after that original epidemic upon contact with Europeans, the most fit survived, and their descendants are today's Native Americans; however, they continue to suffer from contagious diseases at higher rates than the rest of the population.

It has also been said that blacks in the Americas (but especially the US) suffer from hypertension and other heart disease in higher rates compared to other groups, because they are genetically programmed to retain more sodium. According to this theory, in West Africa there were not enough sources of salt, and so in these populations, natural selection favored those who retained sodium (Kiple, 2002:46). During the brutal middle passage to America in slave ships, those who survived were the ones who were better able to retain sodium. And, being slaves in America, the conditions to which they were subjected (work in hot and humid climates) again promoted that natural selection favored those who were able to retain more sodium.

The theory is ingenious, but it is very questionable and unsupported by data. First, it is false that in West Africa there were no sources of salt. Many populations which were

then subject to slavery lived near the coast, and there, they were able to accumulate salt reserves. In fact, populations in West African countries have low blood pressure rates.

In addition, death in voyages on slave ships was not due to diarrhea (associated with dehydration and an inability to retain sodium), but mainly due to respiratory diseases. And if in the warm humid climates plantations, those who survived were the ones that better retained sodium, then one would expect that this applies not only to blacks, but also to whites, i.e., descendants of the masters who lived in those climates.

It is true that there are some diseases afflicting more some racial groups than others. And in general, this is reflected in a considerable difference in the levels of health and life expectancy between racial groups. But again, correlation does not imply causation. We must not rush to postulate that racial constitution is the reason why members of some groups get sicker and live longer than members of other groups. It is necessary to consider other variables.

Some racialists have assumed that the differential in vulnerability to disease and, especially, life expectancy, is caused by racial differences. J.P. Rushton tries to explain this fact in light of his racialist theories. According to his theory of *r* / *k* selection, in populations that evolved in Africa, due to the instability of the habitat, natural selection favored the strategy that consisted in having high fertility rates, lower intelligence, and less care of the young (Rushton, 1995). This strategy focused more on fertility, and in this sense, longevity was not a great advantage, and life cycles of African populations are shorter. That explains why blacks have lower life expectancy and are more vulnerable to many diseases.

Rushton's hypothesis on life expectancy is *ad hoc*. This theory lacks empirical support; it is true that in our society, blacks have a lower life expectancy; but this statistic only establishes a correlation, not a causation (and thus, it does not prove Rushton's hypothesis). There is the possibility that the lower life expectancy of blacks is due to other factors, which Rushton has not noticed.

Another racialist theory aims to locate a genetic origin for the racial disparity in life expectancy, not exactly in physiology, but in intelligence. Biologist Satoshi Kanazawa has documented a correlation between IQ and life expectancy: not surprisingly, sub-Saharan countries, those with lower IQ level, have the lowest life expectancy in the planet (Kanazawa, 2016). According to Kanazawa's theory, in our urban society, outside the context of the African savanna in which the human species originated, the highest levels of health and life expectancy depend on high levels of intelligence, as we face situations which our body is not adapted to, and information processing is required to overcome new obstacles. Lower levels of intelligence promote habits and behaviors that end up being harmful to health. And so, by transitivity, if some racial groups are genetically programmed to have a lower level of intelligence, then they are also genetically programmed to have lower life expectancy.

The data Kanazawa offers is not disputed. Indeed, besides the correlations he has established, it has been documented that there is an inverse correlation between IQ level and consumption of junk food, cigarettes, drugs and alcohol. But again, this just documents a correlation, not causality. Kanazawa assumes that IQ is fixed and determined genetically; this has been subject to intense criticism (Gould, 2006). It is possible to postulate that many social conditions (including racism and oppression) may cause lower levels of intelligence (assuming that IQ is an optimal measure of intelligence,

anyway) of some groups, and that this leads to lower life expectancy. Under this scenario, not genetic constitution, but instead social conditions, cause some groups to have better health indicators and life expectancy than others.

In fact, this is how we must understand the health differential between racial groups. In all societies of the world, groups with lower life expectancy are usually in the lowest position in the social hierarchy. This is due to a wide range of reasons. The poorest groups have less access to health care (whether private or public), and this has a significant impact on health. In addition, the stress to which the oppressed are subject, harmfully affects health.

We must also consider a theory developed by Richard Wilkinson, according to which, the fact that in the African savannah our ancestors did not live the conditions of economic inequality that we have today, gives us difficulties in adapting to contemporary social conditions, and this stressful mismatch generates diseases in us (Wilkinson, 2002). Moreover, Wilkinson argues that this affects not only those in lower positions, but also the economically privileged. The pressure to maintain the status quo and resistance against social climbers, is also harmful to health. Wilkinson documents that countries with better health indices are not exactly the richest, but rather the most egalitarian.

Today most doctors understand diseases as a bio-psycho-social process. Under this paradigm, disease is a multi-causal process, and must be attacked on several fronts, not just the biological one (White, 2005). Above the biological fact of disease, there are added layers of social construction that have a major impact on the way patients develop. A good doctor should not only be aware of the biochemical composition of the drug prescription; he or she should also direct his or her attention to other variables that will have an impact on the patient's treatment: his or her location in the social hierarchy, the patient's willingness to continue the treatment, his or her interpretation of the disease, etc.

We should not ignore the fact that social conditions do have a considerable impact on many diseases. And in that sense, if some racial groups appear to be more vulnerable to a disease, it is not necessarily due to their genes; it can also be due to social position, or in any case, some element of their culture (not registered in genes) that increases the chances of suffering a disease.

To illustrate this argument, let us consider alcoholism among Gypsies. In Spain and other European countries with substantial Roma population, Gypsies' rate of alcoholism is higher than in the rest of the population (Borani, 2002:176). What is this about? We know that alcoholism has a genetic basis: studies with twins and adopted children show a heredity factor of about 27% (Goodwin, 1988). But, is alcoholism among Gypsies due to their genes?

We should not immediately dismiss this possibility, but we should also consider some social factors that may be relevant. Gypsies have been a traditionally excluded social group, and occupy the lowest positions in social hierarchy. We know that lack of opportunities, unemployment, and stress about not getting social promotions; have significant impact on alcohol consumption and eventually, alcohol dependence. Alcoholism is clearly a disease that must be understood in a bio-psycho-social framework.

Some critics worry that this bio-psycho-social paradigm may be abused, and afflicted minorities to pursue victimhood status may easily use it. Critics could claim for example, that alcoholism among Gypsies is due to many other factors, and not just due to

racism in European societies. But even if we accept this view, we are not in need of attempting to explain diseases on the basis of their racial origins. Alcoholism is lowest in Muslim countries: is this due to the genes of Muslim populations, or because of their religion (a cultural factor)? The answer seems obvious.

Let us consider another example. Tuberculosis afflicts a high proportion of Native Americans in Latin American countries (especially the Wayuu of Venezuela and Colombia), largely because in the Native American folk understanding of diseases, sickness is caused by spirits, and in that sense, Native Americans see no need to take prolonged medical treatments (which is very important in the treatment of tuberculosis) (Diaz, 2010). Should we blame racism for this situation? Not necessarily: Native Americans themselves are responsible for not continuing treatment (it could be argued that racism makes Native Americans refuse treatment, but for the sake of argument, let us assume that racism has nothing to do with it). But even in that case, the cause of this high incidence of tuberculosis among indigenous populations would be in their culture, and not in their genes.

Ultimately, doctors do need to take into account what racial group a patient belongs to, but not for the reasons usually cited. The medical ethics principle of justice applies here. There is no doubt that some genetic diseases are more common in some groups than others. However, we should not rush to assume that traditional racial traits have an intrinsic relationship with some genes encoding particular diseases. Sickle cell anemia, for example, has nothing to do with dark skin, but instead, with resistance to malaria.

But the main reason why doctors should be aware of the racial affiliation of their patients is that while human races are mostly a social construct, this social construction has considerable effects on the health of human beings. This does not mean that social ills are social constructs; it means that the social construction of races leads to racism, and racism does have a significant effect on the health of people.

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